From Mad Cows to Humans
THE NEXT GLOBAL PLAGUE

By underestimating the threat and not taking action sooner over the BSE and CJD crises, agricultural and health authorities in Britain and Europe may have unleashed a potentially global and fatal epidemic.

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ACROSS THE SPECIES BARRIER—AND NO CURES IN SIGHT

Speaking from Washington, DC, in October 1997 after hearing of his Nobel Prize win for discovering the role of molecules known as "prions" in the invariably fatal brain illnesses such as "mad cow disease" or bovine spongiform encephalopathy (BSE) in cattle, and Creutzfeldt-Jakob disease (CJD) in humans, Dr Stanley Prusiner from the University of California predicted that the first drug therapy, which would not necessarily be a cure for BSE or CJD, was at least five years away.[1]

At the same time, on the opposite side of the Atlantic, the post-mortem of Chris Warne, a 36-year-old fitness fanatic from Derbyshire, England, revealed that he was the 21st victim of the new variant of CJD which had spread from BSE-infected cattle to humans via the food chain.

Only 18 months earlier, a British House of Commons admission that BSE-infected meat had probably caused the CJD deaths of 10 youthful Britons left the British meat industry in tatters.[2] Since then, the history of BSE has gradually unfolded to reveal a brain-dead imperialism, one which, while blinded by its own arrogant greed to inflate market profits, has treated public and, indeed, world health with gay abandon.

Formerly a rare disease which affected less than one per million in most countries, one worst-case scenario predicts that BSE-infected meat will push the incidence of CJD in humans to claim 10,000 British lives by the year 2000, and a further 10 million by the year 2010. Another predicts that half the British people, some 30 million, will be left brain-dead by CJD. As Chris Warne's mother commented, her son was a health-conscious sportsman, but "*after winning medals in March, by July he couldn't stand on his feet, and by October he was gone*".

A CJD epidemic of these proportions largely defies contemplation, but at the same time it raises important questions of whether nature or human error was responsible for the unprecedented assault of CJD and BSE on humans and animals, and whether the public health implications will, at best, be restricted to Britain and her European cronies, or, at worst, become a global disaster.

Faced with a worldwide boycott of British beef, millions of cattle destined for cremation, and BSE emerging in cattle all across Europe, authorities have disenchantingly persisted with face-saving reassurances, the majority of which are disproven with monotonous regularity.

In keeping with the 1960s to 1985 medical mayhem which turned infertile women and short-statured children into human incubators of CJD with injections of hormones harvested from the pituitary glands of human cadavers, mad-cow globalists view Third World countries as a dumping-ground for BSE-infected meat in their thrust to salvage some cash from the chaos.

Unlike the malignant twists of nature, ranging from bubonic plague through to potato blight, which have killed masses throughout the ages, both the beef and pituitary hormone CJD crises were manmade. Scrapie, the sheep equivalent of BSE and CJD, has been around for more than two centuries. Somewhat differently, human spongiform encephalopathy was unheard of before two German physicians, Creutzfeldt and Jakob, independently reported the initial cases in the 1920s. BSE, too, was unheard of until a decade after cattle began to be fed the protein-rich remains of scrapie-infected sheep to accelerate their growth.

Until the BSE crisis came to a head in 1996, there was no concerted effort to find a diagnostic screening test to identify CJD/BSE infection, and to this day there is no known

medication which can cure or allay the cruelty of human or animal death from the diseases.

In humans, outward warning symptoms only emerge after a prolonged incubation period that, in iatrogenic cases which have occurred as a result of human pituitary growth and infertility hormone injections or contaminated surgical materials, has ranged from as few as two to as many as 40 years.

By that stage, the agent of CJD has already turned the brain into the sponge-like mass that led this group of diseases to be classified as "spongiform slow-virus disorders" in the first instance. Death may be a welcome escape from the involuntary jerking motions which accompany CJD which, while silently eating away at the brain over years, has robbed humans of their every means of communication—the ability to hear, see and speak. Gone, too, is the understanding of written and spoken native language, and with it every scrap of dignity.

Similarly, BSE has no respect for cattle decorum, and a furnace is the fate of confused and trembling animals that the disease has deprived of their own legs on which to stand.

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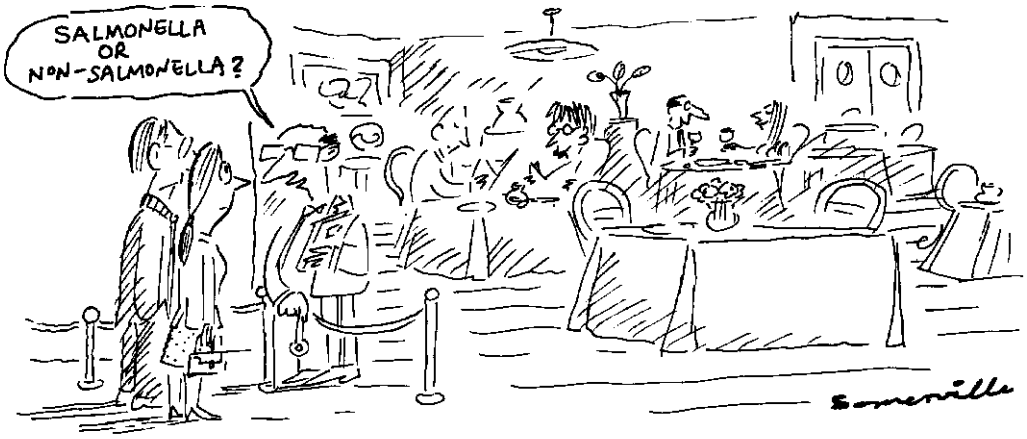
TRACING THE TRANSMISSION ROUTES OF BRAIN DISEASES

The original lesson about the infectious nature of these brain diseases came from a 1934 vaccine catastrophe in the UK which brought scrapie, or "mad sheep disease", to almost 5,000 out of 18,000 lambs within two years of their immunisation against louping-ill virus infection. Tracing back, scientists discovered that the vaccine serum was prepared from a number of lambs whose dams had subsequently developed scrapie, but the significance of scrapie passing vertically from ewes to their lambs, and horizontally from lamb to lamb by virtue of the vaccine injections, was kept from international eyes by a series of egotistical carry-ons which prevented the data from reaching the pages of the scientific literature for a further 15 years.[3]

By then, as the 1950s dawned, mad sheep disease was shown in the United States to jump the species barrier when a scrapie-infected food supplement brought a similar brain illness to farm-raised mink in 1947.[4]

By this stage, the medico-scientific fraternity was intensely preoccupied with another incurable brain illness, kuru, which had reached epidemic proportions amongst the Fore people living in the highlands of New Guinea. Anthropologists from the University of Adelaide unravelled a chain of events to trace the origin of kuru back to the reverent consumption of deceased tribal members' bodies. Kuru was essentially eradicated by New Guinean authorities acting in 1959 on the anthropological clue to outlaw the eating of human flesh. However, the 1976 Nobel Prize went to American scientist Carleton Gajdusek for his experiments demonstrating that injections of kuru brain (1967) and CJD brain (1969) reproduced similar illnesses in chimpanzees.[5] Gajdusek was placed behind bars in 1997 after being found guilty of molesting one the numerous New Guinean youths he has sponsored into the United States over the previous 30 years; however, his research did put an end to ideas that species barriers were an impediment to the spread of this type of disease.

Two neuroscientists, Laura and (the late) Eli Manuelides, from Yale University in the US, went on to illustrate by 1975 that injections of human blood, like injections of brain taken from kuru and CJD victims, transmitted the disease across the species barrier to laboratory animals.[6] Their prophetic, but unheeded, message implied that blood was the vehicle that carried the agent of CJD around the body until it chanced upon an hospitable residence like the brain. This meant that the blood route was the key to the transmission of CJD from a primary host to a secondary host. As distinct from infections such as influenza (which is caused by an airborne virus), but in parallel with AIDS and hepatitis B (which are caused by bloodborne viruses), this indicated that recipients exposed to human pituitary gland hormone injections, or to blood or organ transplants from a donor with CJD, risked becoming secondary CJD hosts once contagious material entered their bloodstreams. Similarly, as the UK Government admitted on 7 October 1997,[1] humans infected with the new variant of CJD coming from BSE-infected meat may spread their CJD via blood donation, thereby hastening the globalisation of the European mad-cow dilemma.



Even as the understanding of spongiform encephalopathy increased, various human pituitary hormone programs in countries such as Australia, France, New Zealand, the United Kingdom and United States were attracting hefty government sponsorships. Few of the programs' stalwarts caught on to the implications of the Manuelides' experiments, and unsuccessful attempts between the years of 1978 and 1982[7]

to filter the CJD agent out of the pituitary hormones being injected into unsuspecting short-statured children and infertile women were left to one of this era's rare visionaries, British scrapie expert Alan Dickinson.

At about the same time, a British Royal Commission on Environmental Pollution in 1979 raised the possibility that the unregulated cycling of protein-rich sheep remains back into animal feed might spread scrapie to cattle, as it had done to farm mink in the US three decades beforehand, via the oral route.

At the same time, too, in the push to meet the insatiable demand for more and more human pituitary hormones, India, the world's second-most-populous country, became a Mecca for pituitary-gland harvests. Literally millions of pituitaries were harvested from cadavers in the subcontinent and sent to government laboratories back in Europe and North America. The promised repayment in kind—namely, with a supply of extracted growth hormone to treat short-statured children in India—simply became another broken imperialist promise, but one which probably accounts for India's enviable position today of remaining a CJD-free country.[8]

By 1985, the first of the fatal legacies of this form of medical madness emerged with four cases of CJD in human pituitary growth hormone-treated children.

Programs were immediately halted in most countries, the notable exception being France where the growth-hormone treatment of children continued—based on the haughty assumption that the purity of the French hormone-extraction process accounted for the absence of a single case of CJD to that point in time. Four years later, in 1989, during which time the number of French children at risk of growth-hormone-related CJD had practically doubled, the first French children fulfilled that tragic legacy. In 1993, those responsible for this travesty were threatened with manslaughter charges. By 1997, France had half of the world's 100-plus cases of pituitary hormone-related CJD.[9]

Although the general elitism of human-pituitary programs restricted this brand of medical madness to North America, Europe and Australasia, Third World children and women did not altogether escape the insanity of applying Frankenstein medicine to social conditions. A medical report in 1991[10] linked the CJD death of a young Brazilian man, like those of five youthful New Zealand men and women,[11] with a childhood treatment involving pituitary growth hormone obtained from the US.

Unfortunately, the fate of women in Mexico City whose breasts were injected with US pituitary hormones in an appalling experiment[12] to increase the volume of milk in lactating mothers (some already pregnant again) will probably never be known.

The opportunity to contain the CJD legacy of pituitary-hormone injections went begging, as blissfully unaware recipients risked spreading their legacy via blood donation. Similarly, the possibility that pituitary-hormone recipients may have transmitted their CJD legacy to their children was totally cast aside.

Oddly, although the entire concept of blood-transfusion-related CJD was publicly dismissed by health authorities, by 1987 all US and New Zealand registered recipients of pituitary growth hormone were advised not to donate blood and organs. It took until 1992 for Australian and British blood banks and transplant programs to follow suit, with the result that the Australian and British general communities were exposed to the risk of secondary CJD transmission for five years longer than their American and New Zealand counterparts.

Somewhat inexplicably, too, despite the theory of blood-transmitted CJD being considered unproven in humans, 1995 and 1996 actions indicate that authorities have finally opened their minds to the public health implications of the Manuelides' experiments. Canadian authorities spent C$15 million in 1995 to withdraw pooled plasma, already in the process of being transfused to thousands across the country, on the grounds that it contained a donation from a man who had subsequently died of CJD.[13] Similarly, in 1996, New Zealand authorities bit the bullet under the weight of public pressure and quarantined blood products which had been contaminated by a donation from a CJD-infected donor;[14] and British blood banks increased their precautionary measures with an extended questioning routine designed to screen out donations from parents, siblings and children of CJD victims.[15]

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British microbiologist Steven Dealler estimates that CJD-infected blood may reach as many as 60,000 recipients each year,[16] but the years-long incubation time preceding CJD symptoms increases the difficulty of linking a blood transfusion recipient's CJD with a donor source. It falls within the realms of possibility that secondary CJD in a transfusion recipient may appear years in advance of the primary CJD in a blood donor, and evidence of blood-transfusion-transmitted CJD was dismissed as anecdotal until 1996, when the case of CJD in a liver transplant recipient was, after the liver donor had been cleared, traced back to a CJD-like illness in one of the blood donors.[17]

MARKETPLACE MADNESS

One year after the first cases of pituitary growth hormone-related CJD in 1985, the first of the animal-protein-fed cattle came down with BSE.[18]

Advisory committees were set up around the world, but none with the foresight to include public health experts trained to weigh policy in terms of both best and worst predictions. Instead, for the next 10 years authorities seized every chance to preserve the reputations and careers of eminent politicians, physicians and scientists, and managed to allay public anxiety by keeping news of their bungles out of the media. Public and animal health ran a very poor second to the market pressures[19] which saw cattle transformed from BSE-free herbivores into BSE-infected carnivores by a nonregulated protein diet. In fact, even as BSE emerged in protein-fed British cattle in 1986, scientific advice that the epidemic could best be contained by compensating farmers for the immediate destruction of the 10,000-odd infected cattle was dismissed because of budgetary concerns.

Following the 1988 ban on scrapie-contaminated animal feed, the BSE epidemic was claimed to be under control. According to authorities, the peak 1992 weekly average of 700 new cases of BSE dropped to 70 cases per week in 1996. At the same time, the notion of control is contradicted by the BSE in some 27,000 cattle born after the 1988 ban. Rather, these figures, together with the 60 per cent of 1996 cases occurring in cattle born post-1988, indicate that pre-feed-regulated cattle have passed BSE onto their calves.

Like the theory of bloodborne CJD in humans, earlier suggestions[20] that the BSE epidemic in cattle was maintained by maternal transmission were dismissed and at times ridiculed, until a 1996 study proved otherwise.[21]

Erring on the side of caution has invariably been forgotten in the brain-dead politicking underpinning the BSE/CJD debacle. As an example, the British Ministry of Agriculture, Fisheries and Food (MAFF) sabotaged a 1990 Brussels ruling designed to prevent the spread of BSE across to the European mainland.[22]

MAFF instead issued civil servants with secret orders to skip the computer-vetting of calves set for the lucrative saleyards of European Union (EU) member countries. As a result, there were no checks to determine whether some two million veal calves sold to the EU between 1990 and 1995 were born to BSE-infected cows or not.

Even the computer tracing of the BSE parentage of some 2,000 cattle sold for foreign breeding after 1990 is untrustworthy, partly because of MAFF's skulduggery, and partly because the sales involved animals that were too young to reveal symptoms of BSE infection—and there is no diagnostic screening test for BSE to establish which cattle are infected and which are free of BSE.

An estimated 700,000 BSE-infected cattle entered the human food chain, chiefly because the animal's slaughter age (usually three years) pre-dated the average age (five years) at which they would show signs of BSE infection.[23] For the same reason, there is simply no way of knowing the number of breeding stock exported to the four corners of the globe before their sire's or dam's BSE was subsequently uncovered.

Britain was not alone in the cover-up of the BSE scandal. In September 1996, the French newspaper *Libération*[24] revealed that a memorandum from French official Gilbert Castille had suggested back in 1990 that Britain ought to be asked *not* to publish its research results, saying, "*it would be better to minimise BSE by practising disinformation*". In fact, rather than ganging up on Britain, Brussels, via Guy Legras, head of the European Commission's agricultural directorate, warned of the financial repercussions from a beef panic and so hushed up news of the BSE situation.

PAYING THE PRICE OF GLOBALISATION

Cattle may not be the only species within the meat industry that is harbouring the BSE/CJD agent in readiness for the food chain. Until March 1996, no restrictions were placed on feeding cattle offal to pigs and hens.[25]

Together with a common practice whereby animal-feed manufacturers share the same equipment to mix both cattle-feed and pig-feed, this approach reflects a glaring ignorance within the agricultural industry about the dangerously infectious nature of diseases such as BSE and CJD.

This background, together with the extreme resistance of BSE and CJD to high temperatures and caustic chemicals that customarily rid instruments and tools of infectious materials, may explain the disproportional excess of CJD infection occurring in the farming community. It also brings the focus back to blood-route-transmitted CJD, and raises the prospect of simple kitchen injuries introducing BSE from infected meat products into the bloodstream of an unsuspecting public.[26]

A worst-case-scenario-sized CJD epidemic will smash rather than stretch every available human resource. European transnationalists, joined in this century by those from the United States, and to a lesser extent Canada and Australia, have widened the gap between developed and developing regions with modern discriminations which transgress the boundaries of human rights, development, environment, nuclear weapons, population, trade and wealth.[27, 28, 29, 30]

Just as medical impropriety, rather than nature, has already destroyed the lives of 100-plus pituitary hormone recipients and their families, agricultural impropriety in the beef and dairy industry, rather than nature, has snuffed out young lives with an atypical but equally cruel form of CJD spread from cattle.

Humans and animals have paid a huge price for the 60-year reign of institutionalised shortsightedness and its underestimated and mistaken grasp of the CJD/BSE contagion. Notions that whitewash the cull of Britain's cattle population to make early inroads into global greenhouse targets[31]—notions like the current sell-off of British meat at record low prices in Asia, and proposals to restock the sacred herds of India and detonate Cambodia's and Afghanistan's landmines with BSE-infected cattle—are barbarous extensions of a brain-dead culture which serve only to hasten the globalisation of the CJD/BSE epidemic.

With mad-cow maniacs intent on adding manmade BSE to the nuclear waste, toxic chemicals and perilous medications which have already turned Third World countries into dumping grounds for developed-world disasters, surely this is proof that little or nothing has been learned from 60 years of "progress" in economics, science and politics. ∞

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